

10/670,065
L/COOL 4/28/07
Search updated.

d his

(FILE 'HOME' ENTERED AT 19:47:25 ON 28 APR 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 19:47:42 ON 28
APR 2007

L1 1995 S MDM AND MACRO?
L2 9 S L1 AND VIMENTIN?
L3 6 DUPLICATE REMOVE L2 (3 DUPLICATES REMOVED)
L4 27 S L1 AND COLI?
L5 14 DUPLICATE REMOVE L4 (13 DUPLICATES REMOVED)
L6 7 S L5 AND PD<2003
L7 17811 S (OKADAIC ACID)
L8 0 S L6 AND L7
L9 343 S GO6983?
L10 0 S L9 AND L6
L11 0 S L9 AND L1
L12 3 S L9 AND VIMENTIN?
L13 1 DUPLICATE REMOVE L12 (2 DUPLICATES REMOVED)
L14 12 S L9 AND BACTE?
L15 642 DUPLICATE REMOVE L1 (1353 DUPLICATES REMOVED)
L16 7 DUPLICATE REMOVE L14 (5 DUPLICATES REMOVED)
L17 1 S L16 AND PD<2003
L18 142 S L7 AND VIMENTIN?
L19 6097 S (SB 203580)
L20 10 S L19 AND VIMENTIN?
L21 10 DUPLICATE REMOVE L20 (0 DUPLICATES REMOVED)
L22 5 S L21 AND PD<2003
L23 12 S L9 AND BACTER?
L24 7 DUPLICATE REMOVE L23 (5 DUPLICATES REMOVED)
L25 1 S L24 AND PD<2003

=>

d his

(FILE 'HOME' ENTERED AT 19:47:25 ON 28 APR 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 19:47:42 ON 28 APR 2007

L1 1995 S MDM AND MACRO?
L2 9 S L1 AND VIMENTIN?
L3 6 DUPLICATE REMOVE L2 (3 DUPLICATES REMOVED)
L4 27 S L1 AND COLI?
L5 14 DUPLICATE REMOVE L4 (13 DUPLICATES REMOVED)
L6 7 S L5 AND PD<2003
L7 17811 S (OKADAIC ACID)
L8 0 S L6 AND L7
L9 343 S GO6983?
L10 0 S L9 AND L6
L11 0 S L9 AND L1
L12 3 S L9 AND VIMENTIN?
L13 1 DUPLICATE REMOVE L12 (2 DUPLICATES REMOVED)
L14 12 S L9 AND BACTE?
L15 642 DUPLICATE REMOVE L1 (1353 DUPLICATES REMOVED)
L16 7 DUPLICATE REMOVE L14 (5 DUPLICATES REMOVED)
L17 1 S L16 AND PD<2003
L18 142 S L7 AND VIMENTIN?
L19 6097 S (SB 203580)
L20 10 S L19 AND VIMENTIN?
L21 10 DUPLICATE REMOVE L20 (0 DUPLICATES REMOVED)
L22 5 S L21 AND PD<2003
L23 12 S L9 AND BACTER?
L24 7 DUPLICATE REMOVE L23 (5 DUPLICATES REMOVED)
L25 1 S L24 AND PD<2003

=>

ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 2001:437639 BIOSIS

DN PREV200100437639

TI Dual actions of *Pasteurella multocida* toxin (Galphag agonist) to promote cardiomyocyte hypertrophy and enhance susceptibility to apoptosis.

AU Sabri, Abdelkarim [Reprint author]; Wilson, Brenda A.; Steinberg, Susan F.

CS Columbia University, New York, NY, USA

SO Journal of Molecular and Cellular Cardiology, (June, 2001) Vol. 33, No. 6, pp. A104. print.

Meeting Info.: XVII ISHR World Congress of the International Society for Heart Research. Winnipeg, Canada. July 06-11, 2001. International Society for Heart Research.

CODEN: JMCDAY. ISSN: 0022-2828.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 12 Sep 2001
Last Updated on STN: 22 Feb 2002

CC General biology - Symposia, transactions and proceedings 00520
Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - General and comparative studies: coenzymes 10802
Cardiovascular system - Physiology and biochemistry 14504
Endocrine - General 17002
Toxicology - General and methods 22501
Physiology and biochemistry of bacteria 31000

IT Major Concepts
Enzymology (Biochemistry and Molecular Biophysics); Toxicology;
Cardiovascular System (Transport and Circulation)

IT Parts, Structures, & Systems of Organisms
cardiac fibroblast: circulatory system; cardiac sarcomere: circulatory system, muscular system; heart: circulatory system

IT Diseases
cardiomyocyte hypertrophy: heart disease, pathogenesis

IT Chemicals & Biochemicals
AKT: phosphorylation; ANF [atrial natriuretic factor]: expression; ERK [extracellular signal-regulated kinase]: activation; Go6983: enzyme inhibitor; Gq protein alpha subunit: expression, regulation; JNK [c-Jun N-terminal kinase]: activation; *Pasteurella multocida* toxin: Gq protein alpha agonist, toxin; epidermal growth factor receptor [EGF receptor]: activation; p38-MAPK [p38-mitogen-activated protein kinase]: activation; phospholipase C: activation; protein kinase C: isoforms, novel, regulation

IT Miscellaneous Descriptors
apoptosis susceptibility modulation; cardiomyocyte apoptosis; Meeting Abstract

ORGN Classifier
Pasteurellaceae 06703
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria; Microorganisms
Organism Name
Pasteurella multocida: toxin source
Taxa Notes
Bacteria, Eubacteria, Microorganisms

RN 133053-19-7 (Go6983)
37333-47-4 (*Pasteurella multocida* toxin)
9001-86-9Q (phospholipase C)
63551-76-8Q (phospholipase C)
141436-78-4 (protein kinase C)
85637-73-6 (ATRIAL NATRIURETIC FACTOR)
142243-02-5 (EXTRACELLULAR SIGNAL-REGULATED KINASE)
155215-87-5 (C-JUN N-TERMINAL KINASE)
165245-96-5 (P38-MITOGEN-ACTIVATED PROTEIN KINASE)

ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 2001:437639 BIOSIS

DN PREV200100437639

TI Dual actions of *Pasteurella multocida* toxin (Galphag agonist) to promote cardiomyocyte hypertrophy and enhance susceptibility to apoptosis.

AU Sabri, Abdelkarim [Reprint author]; Wilson, Brenda A.; Steinberg, Susan F.

CS Columbia University, New York, NY, USA

SO Journal of Molecular and Cellular Cardiology, (June, 2001) Vol. 33, No. 6, pp. A104. print.

Meeting Info.: XVII ISHR World Congress of the International Society for Heart Research. Winnipeg, Canada. July 06-11, 2001. International Society for Heart Research.

CODEN: JMCDAY. ISSN: 0022-2828.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 12 Sep 2001
Last Updated on STN: 22 Feb 2002

CC General biology - Symposia, transactions and proceedings 00520
Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - General and comparative studies: coenzymes 10802
Cardiovascular system - Physiology and biochemistry 14504
Endocrine - General 17002
Toxicology - General and methods 22501
Physiology and biochemistry of bacteria 31000

IT Major Concepts
Enzymology (Biochemistry and Molecular Biophysics); Toxicology;
Cardiovascular System (Transport and Circulation)

IT Parts, Structures, & Systems of Organisms
cardiac fibroblast: circulatory system; cardiac sarcomere: circulatory system, muscular system; heart: circulatory system

IT Diseases
cardiomyocyte hypertrophy: heart disease, pathogenesis

IT Chemicals & Biochemicals
AKT: phosphorylation; ANF [atrial natriuretic factor]: expression; ERK [extracellular signal-regulated kinase]: activation; Go6983: enzyme inhibitor; Gq protein alpha subunit: expression, regulation; JNK [c-Jun N-terminal kinase]: activation; *Pasteurella multocida* toxin: Gq protein alpha agonist, toxin; epidermal growth factor receptor [EGF receptor]: activation; p38-MAPK [p38-mitogen-activated protein kinase]: activation; phospholipase C: activation; protein kinase C: isoforms, novel, regulation

IT Miscellaneous Descriptors
apoptosis susceptibility modulation; cardiomyocyte apoptosis; Meeting Abstract

ORGN Classifier
Pasteurellaceae 06703
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria; Microorganisms
Organism Name
Pasteurella multocida: toxin source
Taxa Notes
Bacteria, Eubacteria, Microorganisms

RN 133053-19-7 (Go6983)
37333-47-4 (*Pasteurella multocida* toxin)
9001-86-9Q (phospholipase C)
63551-76-8Q (phospholipase C)
141436-78-4 (protein kinase C)
85637-73-6 (ATRIAL NATRIURETIC FACTOR)
142243-02-5 (EXTRACELLULAR SIGNAL-REGULATED KINASE)
155215-87-5 (C-JUN N-TERMINAL KINASE)
165245-96-5 (P38-MITOGEN-ACTIVATED PROTEIN KINASE)

ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 2001:437639 BIOSIS

DN PREV200100437639

TI Dual actions of *Pasteurella multocida* toxin (Galphag agonist) to promote cardiomyocyte hypertrophy and enhance susceptibility to apoptosis.

AU Sabri, Abdelkarim [Reprint author]; Wilson, Brenda A.; Steinberg, Susan F.

CS Columbia University, New York, NY, USA

SO Journal of Molecular and Cellular Cardiology, (June, 2001) Vol. 33, No. 6, pp. A104. print.

Meeting Info.: XVII ISHR World Congress of the International Society for Heart Research. Winnipeg, Canada. July 06-11, 2001. International Society for Heart Research.

CODEN: JMCDAY. ISSN: 0022-2828.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 12 Sep 2001
Last Updated on STN: 22 Feb 2002

CC General biology - Symposia, transactions and proceedings 00520
Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - General and comparative studies: coenzymes 10802
Cardiovascular system - Physiology and biochemistry 14504
Endocrine - General 17002
Toxicology - General and methods 22501
Physiology and biochemistry of bacteria 31000

IT Major Concepts
Enzymology (Biochemistry and Molecular Biophysics); Toxicology;
Cardiovascular System (Transport and Circulation)

IT Parts, Structures, & Systems of Organisms
cardiac fibroblast: circulatory system; cardiac sarcomere: circulatory system, muscular system; heart: circulatory system

IT Diseases
cardiomyocyte hypertrophy: heart disease, pathogenesis

IT Chemicals & Biochemicals
AKT: phosphorylation; ANF [atrial natriuretic factor]: expression; ERK [extracellular signal-regulated kinase]: activation; Go6983: enzyme inhibitor; Gq protein alpha subunit: expression, regulation; JNK [c-Jun N-terminal kinase]: activation; *Pasteurella multocida* toxin: Gq protein alpha agonist, toxin; epidermal growth factor receptor [EGF receptor]: activation; p38-MAPK [p38-mitogen-activated protein kinase]: activation; phospholipase C: activation; protein kinase C: isoforms, novel, regulation

IT Miscellaneous Descriptors
apoptosis susceptibility modulation; cardiomyocyte apoptosis; Meeting Abstract

ORGN Classifier
Pasteurellaceae 06703
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria; Microorganisms
Organism Name
Pasteurella multocida: toxin source
Taxa Notes
Bacteria, Eubacteria, Microorganisms

RN 133053-19-7 (Go6983)
37333-47-4 (*Pasteurella multocida* toxin)
9001-86-9Q (phospholipase C)
63551-76-8Q (phospholipase C)
141436-78-4 (protein kinase C)
85637-73-6 (ATRIAL NATRIURETIC FACTOR)
142243-02-5 (EXTRACELLULAR SIGNAL-REGULATED KINASE)
155215-87-5 (C-JUN N-TERMINAL KINASE)
165245-96-5 (P38-MITOGEN-ACTIVATED PROTEIN KINASE)

ANSWER 1 OF 1 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 2001:437639 BIOSIS

DN PREV200100437639

TI Dual actions of *Pasteurella multocida* toxin (Galphag agonist) to promote cardiomyocyte hypertrophy and enhance susceptibility to apoptosis.

AU Sabri, Abdelkarim [Reprint author]; Wilson, Brenda A.; Steinberg, Susan F.

CS Columbia University, New York, NY, USA

SO Journal of Molecular and Cellular Cardiology, (June, 2001) Vol. 33, No. 6, pp. A104. print.

Meeting Info.: XVII ISHR World Congress of the International Society for Heart Research. Winnipeg, Canada. July 06-11, 2001. International Society for Heart Research.

CODEN: JMCDAY. ISSN: 0022-2828.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 12 Sep 2001
Last Updated on STN: 22 Feb 2002

CC General biology - Symposia, transactions and proceedings 00520
Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - General and comparative studies: coenzymes 10802
Cardiovascular system - Physiology and biochemistry 14504
Endocrine - General 17002
Toxicology - General and methods 22501
Physiology and biochemistry of bacteria 31000

IT Major Concepts
Enzymology (Biochemistry and Molecular Biophysics); Toxicology;
Cardiovascular System (Transport and Circulation)

IT Parts, Structures, & Systems of Organisms
cardiac fibroblast: circulatory system; cardiac sarcomere: circulatory system, muscular system; heart: circulatory system

IT Diseases
cardiomyocyte hypertrophy: heart disease, pathogenesis

IT Chemicals & Biochemicals
AKT: phosphorylation; ANF [atrial natriuretic factor]: expression; ERK [extracellular signal-regulated kinase]: activation; Go6983: enzyme inhibitor; Gq protein alpha subunit: expression, regulation; JNK [c-Jun N-terminal kinase]: activation; *Pasteurella multocida* toxin: Gq protein alpha agonist, toxin; epidermal growth factor receptor [EGF receptor]: activation; p38-MAPK [p38-mitogen-activated protein kinase]: activation; phospholipase C: activation; protein kinase C: isoforms, novel, regulation

IT Miscellaneous Descriptors
apoptosis susceptibility modulation; cardiomyocyte apoptosis; Meeting Abstract

ORGN Classifier
Pasteurellaceae 06703
Super Taxa
Facultatively Anaerobic Gram-Negative Rods; Eubacteria;
Bacteria; Microorganisms
Organism Name
Pasteurella multocida: toxin source
Taxa Notes
Bacteria, Eubacteria, Microorganisms

RN 133053-19-7 (Go6983)
37333-47-4 (*Pasteurella multocida* toxin)
9001-86-9Q (phospholipase C)
63551-76-8Q (phospholipase C)
141436-78-4 (protein kinase C)
85637-73-6 (ATRIAL NATRIURETIC FACTOR)
142243-02-5 (EXTRACELLULAR SIGNAL-REGULATED KINASE)
155215-87-5 (C-JUN N-TERMINAL KINASE)
165245-96-5 (P38-MITOGEN-ACTIVATED PROTEIN KINASE)

THE STUDY ON THE RELATIONSHIP BETWEEN HUMAN CYTOMEGALOVIRUS INFECTION AND ATHEROSCLEROSIS DEVELOPMENT

Chen Ruizhen, Yang Yingzhen, Fu Weiguo, Wang Yuqi, Ge Junbo. (Key Laboratory of Viral Heart Diseases of Ministry of Public Health; Shanghai Institute of Cardiovascular Diseases; Zhongshan Hospital, Fudan University, Shanghai 200032 China)

To investigate the viral etiological factor of Atherosclerosis (AS). Artery vascular tissue specimens derived from 75 patients with AS were studied for detection of human cytomegalovirus (HCMV) immediately early (IE) and later (L) gene fragments by polymerase chain reaction (PCR) and in situ hybridization; sera collected from the same patients were examined for HCMV specific IgG and IgM by enzyme-linked immunosorbent assay (ELISA). Twenty two normal arterial tissues and sera were used as controls. The positive rate of HCMV L and IE gene fragments were significantly higher in AS patients than those in controls. HCMV DNA were mainly observed in nucleus of endothelial cells, muscularis under intima and smooth muscle of media of AS, rarely found in controls. There are some relationship between HCMV and AS; Artery itself maybe the site of HCMV latency. Meanwhile, Higher levels of HCMV-sIgG and sIgM were found to be associated virus, indicating that a periodically active latent infection or a continuously active infection is presented in AS patients.

INVOLVEMENT OF 1,2-DIACYLGLYCEROL IN CARDIAC HYPERTROPHY OF THE JUVENILE VISCERAL STEATOSIS MICE WITH SYSTEMIC CARNITINE DEFICIENCY

Yoshihiro Saburi, Hiroki Kaniya, Kazunori Hayashi, Hideo Matsui, Kazji Okumura

Background: The juvenile visceral steatosis (JVS) mouse, an animal model of primary systemic carnitine deficiency, is known to develop cardiac hypertrophy. This study was performed as to whether cardiac hypertrophy in JVS mice is associated with the quantitative and qualitative changes of 1,2-diacylglycerol (1,2-DAG), an activator of protein kinase C.

Methods: Controls and JVS mice were evaluated at 4 weeks (developing hypertrophy stage) and 8 weeks (established hypertrophy stage) of age. Myocardial 1,2-DAG level was measured by FID-TLC method and its fatty acid composition was analysed by gas chromatography.

Results: The heart weight/body weight ratio was 2.5-fold higher and left ventricle wall thickness was 1.4-fold higher in JVS mice at 8 weeks compared with those of controls ($p < 0.01$, respectively). Myocardial 1,2-DAG level at 4 weeks was already elevated in JVS mice (1023 vs. 1366 ng/dry wt, $p = 0.07$). At 8 weeks, the 1,2-DAG level was remarkably increased in JVS mice compared with that in controls (819 vs. 2053 ng/dry wt, $p < 0.01$). Fatty acids of 18:1 and 18:2 were significantly elevated in hearts of JVS mice both at 4 and 8 weeks compared with those of controls ($P < 0.01$, respectively).

Conclusion: Increase in distinct 1,2-DAG species containing 18:1 and 18:2 fatty acids might be involved in the pathogenesis of cardiac hypertrophy in JVS with systemic carnitine deficiency.

DUAL ACTIONS OF PASTEURELLA MULTOCIDA TOXIN (Gq α AGONIST) TO PROMOTE CARDIOMYOCYTE HYPERTROPHY AND ENHANCE SUSCEPTIBILITY TO APOPTOSIS

Abdelkarim Sabri, Brenda A. Wilson & Susan F. Steinberg
Columbia University, NY & Univ IL at Urbana-Champaign

Previous attempts to delineate the consequences of Gq protein α subunit activation in cardiomyocytes relied largely on molecular strategies in cultures or transgenic mice. Modest levels of WT Gq α overexpression induce stable cardiac hypertrophy, whereas intense Gq α stimulation (with high levels of WT Gq α or the constitutively activated mutant) induces cardiomyocyte apoptosis. The precise mechanism(s) whereby traditional targets of Gq α that induce hypertrophy also trigger cardiomyocyte apoptosis is not obvious and is explored with recombinant *Pasteurella multocida* toxin (rPMT, a Gq α agonist). Chronic stimulation with rPMT leads to cardiomyocyte enlargement, sarcomeric organization, and increased ANF expression in association with activation of phospholipase C, novel protein kinase C (PKC) isoforms, ERK, and (to a lesser extent) JNK/p38-MAPK. rPMT stimulates the ERK cascade via epidermal growth factor (EGF) receptor transactivation in cardiac fibroblasts, but not in cardiomyocytes. Surprisingly, rPMT (or PKC activation by phorbol ester) decreases basal AKT phosphorylation and prevents AKT phosphorylation by EGF. The rPMT-dependent decrease in AKT phosphorylation is abrogated by Go6983 (PKC inhibitor) and is functionally significant; cardiomyocyte apoptosis is augmented in rPMT-treated cultures. These results link nPKC isoform activation by Gq α to reduced AKT phosphorylation, impaired AKT stimulation by survival pathways, and enhanced apoptosis susceptibility. AKT inhibition by PKC is predicted to contribute to the transition from hypertrophy to heart failure and represent a target for therapy.

ENDOTHELIAL CELL DYSFUNCTION-KEY FACTOR IN ATHEROGENESIS AND ITS REVERSAL (LABORATORY & CLINICAL STUDY)

G S Sainani, Manisha Sawhney
Jaslok Hosp. & Research Centre, Mumbai (India)

In macrophage cell cultures and DNA studies, we recorded morphological changes and DNA damage caused by hydrogen peroxide exposure. In another experiment native LDL was converted to oxidised LDL by exposure to cigarette smoke extract. In another experiment, interaction of altered macrophages and OX-LDL resulted in foam cells. However pre-treatment with anti-oxidants-superoxide dismutase (SOD), nitric oxide (NO), glutathione peroxidase (Gpx), vitamins E, C, β -carotene in all above experiments reversed partially/completely oxidative damage of macrophages, DNA, LDL and prevented foam cell formation. Clinical study included 100 documented cases of coronary artery disease (CAD) (50-angiographically proved, 50 acute-myocardial infarction patients and 100 healthy controls (angiographically negative). We estimated oxidative stress (thiobarbituric acid reactive substances and conjugated diene levels) and antioxidant levels (SOD, NO, Gpx, vitamins E, C, β -carotene, selenium) in all. Anti-oxidant levels were significantly low in CAD group. Low NO levels in CAD reflect endothelial cell dysfunction (ECD). Coronary artery lesions improved with treatment. We conclude that ECD is the key factor in CAD and with timely treatment, ECD and coronary artery blocks can be reversed to variable extent.

10/670, 065
L/cook 4/28/07
Search updated

d his

(FILE 'HOME' ENTERED AT 20:44:32 ON 28 APR 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 20:45:00 ON 28
APR 2007

L1 44 S (VIMENTIN KINASE)
L2 42232 S VIMENTIN?
L3 4643 S L2 AND MONOCLONAL?
L4 832 S L3 AND POLYCLONAL?
L5 4 S L4 AND (OKADAIC ACID)
L6 1 DUPLICATE REMOVE L5 (3 DUPLICATES REMOVED)
L7 0 S L2 AND ANTISENSE?
L8 237 S L2 AND ANTISENSE?
L9 38 S L8 AND VECTOR?
L10 22 S L9 AND PD<2003
L11 25687 S SIRNA
L12 81 S L11 AND L2
L13 7 S L12 AND L8
L14 0 S L13 AND PD<2003
L15 8 S L10 AND RNA?
L16 12 S L9 AND RNA?
L17 1832 S (THIOL PROTEINASE)
L18 43 S (NEUTRAL THIOL PROTEINASE)
L19 15 S L18 AND L2
L20 5 DUPLICATE REMOVE L19 (10 DUPLICATES REMOVED)
L21 5 S L20 AND PD<2003

d his

(FILE 'HOME' ENTERED AT 20:44:32 ON 28 APR 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 20:45:00 ON 28 APR 2007

```
L1      44 S (VIMENTIN KINASE)
L2      42232 S VIMENTIN?
L3      4643 S L2 AND MONOCLONAL?
L4      832 S L3 AND POLYCLONAL?
L5      4 S L4 AND (OKADAIC ACID)
L6      1 DUPLICATE REMOVE L5 (3 DUPLICATES REMOVED)
L7      0 S L2 AND ANTISENSE?
L8      237 S L2 AND ANTISENSE?
L9      38 S L8 AND VECTOR?
L10     22 S L9 AND PD<2003
L11     25687 S SIRNA
L12     81 S L11 AND L2
L13     7 S L12 AND L8
L14     0 S L13 AND PD<2003
L15     8 S L10 AND RNA?
L16     12 S L9 AND RNA?
L17     1832 S (THIOL PROTEINASE)
L18     43 S (NEUTRAL THIOL PROTEINASE)
L19     15 S L18 AND L2
L20     5 DUPLICATE REMOVE L19 (10 DUPLICATES REMOVED)
L21     5 S L20 AND PD<2003
```

d his

(FILE 'HOME' ENTERED AT 20:44:32 ON 28 APR 2007)

FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, JAPIO' ENTERED AT 20:45:00 ON 28 APR 2007

```
L1      44 S (VIMENTIN KINASE)
L2      42232 S VIMENTIN?
L3      4643 S L2 AND MONOCLONAL?
L4      832 S L3 AND POLYCLONAL?
L5      4 S L4 AND (OKADAIC ACID)
L6      1 DUPLICATE REMOVE L5 (3 DUPLICATES REMOVED)
L7      0 S L2 AND ANTISENSE?
L8      237 S L2 AND ANTISENSE?
L9      38 S L8 AND VECTOR?
L10     22 S L9 AND PD<2003
L11     25687 S SIRNA
L12     81 S L11 AND L2
L13     7 S L12 AND L8
L14     0 S L13 AND PD<2003
L15     8 S L10 AND RNA?
L16     12 S L9 AND RNA?
L17     1832 S (THIOL PROTEINASE)
L18     43 S (NEUTRAL THIOL PROTEINASE)
L19     15 S L18 AND L2
L20     5 DUPLICATE REMOVE L19 (10 DUPLICATES REMOVED)
L21     5 S L20 AND PD<2003
```

ANSWER 1 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1988:480120 BIOSIS

DN PREV198886111430; BA86:111430

TI DIFFERENTIAL SENSITIVITY OF VIMENTIN AND NUCLEAR LAMINS FROM
EHRlich ASCITES TUMOR CELLS TOWARD CALCIUM-ACTIVATED NEUTRAL
THIOL PROTEINASE.

AU TRAUB P [Reprint author]; SCHERBARTH A; WILLINGALE-THEUNE J;
PAULIN-LEVASSEUR M; SHOEMAN R

CS MAX-PLANCK-INST ZELLBIOL, ROSENHOF, D-6802 LADENBURG, W GER

SO European Journal of Cell Biology, (1988) Vol. 46, No. 3, pp.
478-490.
CODEN: EJCBDN. ISSN: 0171-9335.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 1 Nov 1988
Last Updated on STN: 1 Nov 1988

AB A comparative study of the susceptibility of vimentin and
nuclear lamins from cultured Ehrlich ascites tumor (EAT) cells to
degradation of Ca²⁺-activated neutral thiol
proteinase (calpain) has been undertaken. While pure
vimentin was degraded very quickly at physiological ionic strength
by putrified calpain, isolated lamin B was digested comparatively slowly
and purified lamins A/C were fairly resistant to proteolytic degradation.
Similar digestion patterns were obtained from vimentin and lamin
B with intermediary breakdown products close in size to the corresponding
 α -helical rod domains. The exclude the possibility that the low
susceptibility of isolated lamins to Ca²⁺-dependent proteolytic
degradation was due to irreversible denaturation during their isolation
and purification, Triton cytoskeletons were prepared and their nuclear
lamina as well as vimentin filaments were exposed to relatively
large quantities of purified calpain. Under these conditions, not only
vimentin filaments but also lamins A and B were digested while
lamin C remained intact to a high degree. The major breakdown products of
vimentin and lamins were identified as polypeptides which were 35
to 45 amino acids longer than the corresponding α -helical rod
domains. Most of the vimentin-derived material and all high
molecular weight polypeptides originating from lamins remained associated
with the Triton cytoskeletons as demonstrated by sodium dodecyl sulfate
polyacrylamide gel electrophoresis in conjunction with immunoblotting.
Indirect immunofluorescence and electron microscope analysis of the
calpain-digested Triton cytoskeletons revealed that they still contained a
laminalike structure around the nuclear chromatin and numerous
structurally altered intermediate filaments in the cytoplasmic remnant,
although all vimentin had been degraded with the formation of
40/41 kDa polypeptides as major digestion products. In untreated Triton
cytoskeletons, the vimentin filaments seemed to be in direct
physical contact with the nuclear lamina, whereas in digested Triton
cytoskeletons there was a distinct gap between structurally altered
filaments and the nuclear surface. This shows that vimentin
filaments and the nuclear lamina are differently susceptible to
degradation by calpain under certain ionic conditions and suggests that
both filamentous structures are intimately associated with each other.
Moreover, the large differences in isoelectric point between lamins A/C
and lamin B as well as the high degree of microheterogeneity of lamins
isolated from proliferating EAT cells might result primarily from
structural differences in the α -helical rod domains of these
karyoskeletal proteins.

CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - Chemical and physical 10806
Metabolism - Proteins, peptides and amino acids 13012

IT Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Enzymology

ANSWER 1 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1988:480120 BIOSIS

DN PREV198886111430; BA86:111430

TI DIFFERENTIAL SENSITIVITY OF VIMENTIN AND NUCLEAR LAMINS FROM
EHRlich ASCITES TUMOR CELLS TOWARD CALCIUM-ACTIVATED NEUTRAL
THIOL PROTEINASE.

AU TRAUB P [Reprint author]; SCHERBARTH A; WILLINGALE-THEUNE J;
PAULIN-LEVASSEUR M; SHOEMAN R

CS MAX-PLANCK-INST ZELLBIOL, ROSENHOF, D-6802 LADENBURG, W GER

SO European Journal of Cell Biology, (1988) Vol. 46, No. 3, pp.
478-490.
CODEN: EJCBND. ISSN: 0171-9335.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 1 Nov 1988
Last Updated on STN: 1 Nov 1988

AB A comparative study of the susceptibility of vimentin and
nuclear lamins from cultured Ehrlich ascites tumor (EAT) cells to
degradation of Ca²⁺-activated neutral thiol
proteinase (calpain) has been undertaken. While pure
vimentin was degraded very quickly at physiological ionic strength
by putrified calpain, isolated lamin B was digested comparatively slowly
and purified lamins A/C were fairly resistant to proteolytic degradation.
Similar digestion patterns were obtained from vimentin and lamin
B with intermediary breakdown products close in size to the corresponding
 α -helical rod domains. The exclude the possibility that the low
susceptibility of isolated lamins to Ca²⁺-dependent proteolytic
degradation was due to irreversible denaturation during their isolation
and purification, Triton cytoskeletons were prepared and their nuclear
lamina as well as vimentin filaments were exposed to relatively
large quantities of purified calpain. Under these conditions, not only
vimentin filaments but also lamins A and B were digested while
lamin C remained intact to a high degree. The major breakdown products of
vimentin and lamins were identified as polypeptides which were 35
to 45 amino acids longer than the corresponding α -helical rod
domains. Most of the vimentin-derived material and all high
molecular weight polypeptides originating from lamins remained associated
with the Triton cytoskeletons as demonstrated by sodium dodecyl sulfate
polyacrylamide gel electrophoresis in conjunction with immunoblotting.
Indirect immunofluorescence and electron microscope analysis of the
calpain-digested Triton cytoskeletons revealed that they still contained a
laminalike structure around the nuclear chromatin and numerous
structurally altered intermediate filaments in the cytoplasmic remnant,
although all vimentin had been degraded with the formation of
40/41 kDa polypeptides as major digestion products. In untreated Triton
cytoskeletons, the vimentin filaments seemed to be in direct
physical contact with the nuclear lamina, whereas in digested Triton
cytoskeletons there was a distinct gap between structurally altered
filaments and the nuclear surface. This shows that vimentin
filaments and the nuclear lamina are differently susceptible to
degradation by calpain under certain ionic conditions and suggests that
both filamentous structures are intimately associated with each other.
Moreover, the large differences in isoelectric point between lamins A/C
and lamin B as well as the high degree of microheterogeneity of lamins
isolated from proliferating EAT cells might result primarily from
structural differences in the α -helical rod domains of these
karyoskeletal proteins.

CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - Chemical and physical 10806
Metabolism - Proteins, peptides and amino acids 13012

IT Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Enzymology

ANSWER 1 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1988:480120 BIOSIS

DN PREV198886111430; BA86:111430

TI DIFFERENTIAL SENSITIVITY OF VIMENTIN AND NUCLEAR LAMINS FROM
EHRlich ASCITES TUMOR CELLS TOWARD CALCIUM-ACTIVATED NEUTRAL
THIOL PROTEINASE.

AU TRAUB P [Reprint author]; SCHERBARTH A; WILLINGALE-THEUNE J;
PAULIN-LEVASSEUR M; SHOEMAN R

CS MAX-PLANCK-INST ZELLBIOL, ROSENHOF, D-6802 LADENBURG, W GER

SO European Journal of Cell Biology, (1988) Vol. 46, No. 3, pp.
478-490.
CODEN: EJCBDN. ISSN: 0171-9335.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 1 Nov 1988
Last Updated on STN: 1 Nov 1988

AB A comparative study of the susceptibility of vimentin and
nuclear lamins from cultured Ehrlich ascites tumor (EAT) cells to
degradation of Ca²⁺-activated neutral thiol
proteinase (calpain) has been undertaken. While pure
vimentin was degraded very quickly at physiological ionic strength
by putrified calpain, isolated lamin B was digested comparatively slowly
and purified lamins A/C were fairly resistant to proteolytic degradation.
Similar digestion patterns were obtained from vimentin and lamin
B with intermediary breakdown products close in size to the corresponding
 α -helical rod domains. The exclude the possibility that the low
susceptibility of isolated lamins to Ca²⁺-dependent proteolytic
degradation was due to irreversible denaturation during their isolation
and purification, Triton cytoskeletons were prepared and their nuclear
lamina as well as vimentin filaments were exposed to relatively
large quantities of purified calpain. Under these conditions, not only
vimentin filaments but also lamins A and B were digested while
lamin C remained intact to a high degree. The major breakdown products of
vimentin and lamins were identified as polypeptides which were 35
to 45 amino acids longer than the corresponding α -helical rod
domains. Most of the vimentin-derived material and all high
molecular weight polypeptides originating from lamins remained associated
with the Triton cytoskeletons as demonstrated by sodium dodecyl sulfate
polyacrylamide gel electrophoresis in conjunction with immunoblotting.
Indirect immunofluorescence and electron microscope analysis of the
calpain-digested Triton cytoskeletons revealed that they still contained a
laminalike structure around the nuclear chromatin and numerous
structurally altered intermediate filaments in the cytoplasmic remnant,
although all vimentin had been degraded with the formation of
40/41 kDa polypeptides as major digestion products. In untreated Triton
cytoskeletons, the vimentin filaments seemed to be in direct
physical contact with the nuclear lamina, whereas in digested Triton
cytoskeletons there was a distinct gap between structurally altered
filaments and the nuclear surface. This shows that vimentin
filaments and the nuclear lamina are differently susceptible to
degradation by calpain under certain ionic conditions and suggests that
both filamentous structures are intimately associated with each other.
Moreover, the large differences in isoelectric point between lamins A/C
and lamin B as well as the high degree of microheterogeneity of lamins
isolated from proliferating EAT cells might result primarily from
structural differences in the α -helical rod domains of these
karyoskeletal proteins.

CC Cytology - Animal 02506
Biochemistry studies - Proteins, peptides and amino acids 10064
Enzymes - Chemical and physical 10806
Metabolism - Proteins, peptides and amino acids 13012

IT Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Enzymology

(Biochemistry and Molecular Biophysics)
IT Miscellaneous Descriptors
MOUSE CALPAIN INTERMEDIATE FILAMENT CYTOSKELETON CELL CYCLE
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,
Rodents, Vertebrates
RN 7440-70-2 (CALCIUM)
37353-41-6 (THIOL PROTEINASE)
78990-62-2 (CALPAIN)

(Biochemistry and Molecular Biophysics)
IT Miscellaneous Descriptors
MOUSE CALPAIN INTERMEDIATE FILAMENT CYTOSKELETON CELL CYCLE
ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,
Rodents, Vertebrates
RN 7440-70-2 (CALCIUM)
37353-41-6 (THIOL PROTEINASE)
78990-62-2 (CALPAIN)

(Biochemistry and Molecular Biophysics)

IT Miscellaneous Descriptors

MOUSE CALPAIN INTERMEDIATE FILAMENT CYTOSKELETON CELL CYCLE

ORGN Classifier

Muridae 86375

Super Taxa

Rodentia; Mammalia; Vertebrata; Chordata; Animalia

Taxa Notes

Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,

Rodents, Vertebrates

RN 7440-70-2 (CALCIUM)

37353-41-6 (THIOL PROTEINASE)

78990-62-2 (CALPAIN)

ANSWER 3 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1987:104624 BIOSIS

DN PREV198783053602; BA83:53602

TI PROTEIN-CHEMICAL IDENTIFICATION OF THE MAJOR CLEAVAGE SITE OF THE CALCIUM
PROTEINASE ON MURINE VIMENTIN THE MESENCHYMAL INTERMEDIATE
FILAMENT PROTEIN.

AU FISCHER S [Reprint author]; VANDEKERCKHOVE J; AMPE C; TRAUB P; WEBER K

CS MAX-PLANCK-INST BIOPHYS CHEM, POSTFACH 2841, D-3400 GOETTINGEN, W GER

SO Biological Chemistry Hoppe-Seyler, (1986) Vol. 367, No. 11, pp.
1147-1152.
CODEN: BCHSEI. ISSN: 0177-3593.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 26 Feb 1987
Last Updated on STN: 26 Feb 1987

AB Neutral thiol proteinases (calpains),
activated by calcium are involved in the intracellular turnover of
intermediate filaments but the precise position of the cleavage points has
remained unknown. Here we identify by direct sequence analysis the major
cleavage sites found when murine vimentin is digested by limited
proteolysis in vitro with calpain purified from porcine kidney. Contrary
to some previous suggestions, no absolute sequence specificity could be
detected although 10 specific sites have been identified. This result is
in line with cDNA derived amino-acid sequence of a calpain, which pointed
to a similarity of the catalytic site with the active sites in papain,
cathepsin and actinidin. However, all major cleavage sites are located
within regions of the vimentin molecule, which in current models
of intermediate filament structure are thought to be non-helical: the
amino-terminal headpiece, the carboxyl-terminal tail-piece and the spacer
separating the two major coiled-coil domains. The sequence information
about the cleavage sites was extended to provide the amino-terminal 119
residues of murine vimentin.

CC Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Minerals 10069
Biophysics - Biocybernetics 10515
Enzymes - General and comparative studies: coenzymes 10802
Enzymes - Chemical and physical 10806

IT Major Concepts
Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and
Molecular Biophysics)

IT Miscellaneous Descriptors
COMPLEMENTARY DNA-DERIVED AMINO ACID SEQUENCE

ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,
Rodents, Vertebrates

ANSWER 3 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1987:104624 BIOSIS

DN PREV198783053602; BA83:53602

TI PROTEIN-CHEMICAL IDENTIFICATION OF THE MAJOR CLEAVAGE SITE OF THE CALCIUM
PROTEINASE ON MURINE VIMENTIN THE MESENCHYMAL INTERMEDIATE
FILAMENT PROTEIN.

AU FISCHER S [Reprint author]; VANDEKERCKHOVE J; AMPE C; TRAUB P; WEBER K

CS MAX-PLANCK-INST BIOPHYS CHEM, POSTFACH 2841, D-3400 GOETTINGEN, W GER

SO Biological Chemistry Hoppe-Seyler, (1986) Vol. 367, No. 11, pp.
1147-1152.
CODEN: BCHSEI. ISSN: 0177-3593.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 26 Feb 1987
Last Updated on STN: 26 Feb 1987

AB Neutral thiol proteinases (calpains),
activated by calcium are involved in the intracellular turnover of
intermediate filaments but the precise position of the cleavage points has
remained unknown. Here we identify by direct sequence analysis the major
cleavage sites found when murine vimentin is digested by limited
proteolysis in vitro with calpain purified from porcine kidney. Contrary
to some previous suggestions, no absolute sequence specificity could be
detected although 10 specific sites have been identified. This result is
in line with cDNA derived amino-acid sequence of a calpain, which pointed
to a similarity of the catalytic site with the active sites in papain,
cathepsin and actinidin. However, all major cleavage sites are located
within regions of the vimentin molecule, which in current models
of intermediate filament structure are thought to be non-helical: the
amino-terminal headpiece, the carboxyl-terminal tail-piece and the spacer
separating the two major coiled-coil domains. The sequence information
about the cleavage sites was extended to provide the amino-terminal 119
residues of murine vimentin.

CC Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Minerals 10069
Biophysics - Biocybernetics 10515
Enzymes - General and comparative studies: coenzymes 10802
Enzymes - Chemical and physical 10806

IT Major Concepts
Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and
Molecular Biophysics)

IT Miscellaneous Descriptors
COMPLEMENTARY DNA-DERIVED AMINO ACID SEQUENCE

ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,
Rodents, Vertebrates

ANSWER 3 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1987:104624 BIOSIS

DN PREV198783053602; BA83:53602

TI PROTEIN-CHEMICAL IDENTIFICATION OF THE MAJOR CLEAVAGE SITE OF THE CALCIUM
PROTEINASE ON MURINE VIMENTIN THE MESENCHYMAL INTERMEDIATE
FILAMENT PROTEIN.

AU FISCHER S [Reprint author]; VANDEKERCKHOVE J; AMPE C; TRAUB P; WEBER K

CS MAX-PLANCK-INST BIOPHYS CHEM, POSTFACH 2841, D-3400 GOETTINGEN, W GER

SO Biological Chemistry Hoppe-Seyler, (1986) Vol. 367, No. 11, pp.
1147-1152.
CODEN: BCHSEI. ISSN: 0177-3593.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 26 Feb 1987
Last Updated on STN: 26 Feb 1987

AB Neutral thiol proteinases (calpains),
activated by calcium are involved in the intracellular turnover of
intermediate filaments but the precise position of the cleavage points has
remained unknown. Here we identify by direct sequence analysis the major
cleavage sites found when murine vimentin is digested by limited
proteolysis in vitro with calpain purified from porcine kidney. Contrary
to some previous suggestions, no absolute sequence specificity could be
detected although 10 specific sites have been identified. This result is
in line with cDNA derived amino-acid sequence of a calpain, which pointed
to a similarity of the catalytic site with the active sites in papain,
cathepsin and actinidin. However, all major cleavage sites are located
within regions of the vimentin molecule, which in current models
of intermediate filament structure are thought to be non-helical: the
amino-terminal headpiece, the carboxyl-terminal tail-piece and the spacer
separating the two major coiled-coil domains. The sequence information
about the cleavage sites was extended to provide the amino-terminal 119
residues of murine vimentin.

CC Biochemistry studies - Proteins, peptides and amino acids 10064
Biochemistry studies - Minerals 10069
Biophysics - Biocybernetics 10515
Enzymes - General and comparative studies: coenzymes 10802
Enzymes - Chemical and physical 10806

IT Major Concepts
Biochemistry and Molecular Biophysics; Enzymology (Biochemistry and
Molecular Biophysics)

IT Miscellaneous Descriptors
COMPLEMENTARY DNA-DERIVED AMINO ACID SEQUENCE

ORGN Classifier
Muridae 86375
Super Taxa
Rodentia; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Chordates, Mammals, Nonhuman Vertebrates, Nonhuman Mammals,
Rodents, Vertebrates

ANSWER 4 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1986:357972 BIOSIS

DN PREV198682062446; BA82:62446

TI EFFICIENT DEGRADATION IN-VITRO OF ALL INTERMEDIATE FILAMENT SUBUNIT
PROTEINS BY THE CALCIUM-ACTIVATED NEUTRAL THIOL
PROTEINASE FROM EHRLICH ASCITES TUMOR CELLS AND PORCINE KIDNEY.

AU VORGAS C E [Reprint author]; TRAUB P

CS MAX-PLANCK-INST ZELLBIOL, ROSENHOF, D-6802 LADENBURG/HEIDELBERG, FRG

SO Bioscience Reports, (1986) Vol. 6, No. 1, pp. 57-64.
CODEN: BRPTDT. ISSN: 0144-8463.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 6 Sep 1986
Last Updated on STN: 6 Sep 1986

AB Vimentin, desmin, glial fibrillary acidic protein, neurofilament
triplet proteins, and a mixture of cytokeratins were digested with
Ca²⁺-activated neutral thiol proteinase
isolated from Ehrlich ascites tumor (EAT) cells and porcine kidney. All
intermediate filament proteins were degraded by the proteinase, although
with different rates and Ca²⁺ optima. These results are in part at
variance with our previous statement that the Ca²⁺-activated proteinase
from EAT cells is specific for vimentin and desmin.

CC Cytology - Animal 02506
Biochemistry studies - Minerals 10069
Enzymes - Physiological studies 10808
Metabolism - Proteins, peptides and amino acids 13012
Urinary system - Physiology and biochemistry 15504
Neoplasms - Neoplastic cell lines 24005
In vitro cellular and subcellular studies 32600

IT Major Concepts
Cell Biology; Enzymology (Biochemistry and Molecular Biophysics);
Metabolism

IT Miscellaneous Descriptors
VIMENTIN DESMIN GLIAL FIBRILLARY ACIDIC PROTEIN NEUROFILAMENT
TRIPLET PROTEINS CYTOKERATINS

ORGN Classifier
Suidae 85740
Super Taxa
Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Artiodactyls, Chordates, Mammals, Nonhuman Vertebrates,
Nonhuman Mammals, Vertebrates

RN 7440-70-2 (CALCIUM)
37353-41-6 (THIOL PROTEINASE)

ANSWER 4 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1986:357972 BIOSIS

DN PREV198682062446; BA82:62446

TI EFFICIENT DEGRADATION IN-VITRO OF ALL INTERMEDIATE FILAMENT SUBUNIT
PROTEINS BY THE CALCIUM-ACTIVATED NEUTRAL THIOL
PROTEINASE FROM EHRLICH ASCITES TUMOR CELLS AND PORCINE KIDNEY.

AU VORGAS C E [Reprint author]; TRAUB P

CS MAX-PLANCK-INST ZELLBIOL, ROSENHOF, D-6802 LADENBURG/HEIDELBERG, FRG

SO Bioscience Reports, (1986) Vol. 6, No. 1, pp. 57-64.
CODEN: BRPTDT. ISSN: 0144-8463.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 6 Sep 1986
Last Updated on STN: 6 Sep 1986

AB Vimentin, desmin, glial fibrillary acidic protein, neurofilament
triplet proteins, and a mixture of cytokeratins were digested with
Ca²⁺-activated neutral thiol proteinase
isolated from Ehrlich ascites tumor (EAT) cells and porcine kidney. All
intermediate filament proteins were degraded by the proteinase, although
with different rates and Ca²⁺ optima. These results are in part at
variance with our previous statement that the Ca²⁺-activated proteinase
from EAT cells is specific for vimentin and desmin.

CC Cytology - Animal 02506
Biochemistry studies - Minerals 10069
Enzymes - Physiological studies 10808
Metabolism - Proteins, peptides and amino acids 13012
Urinary system - Physiology and biochemistry 15504
Neoplasms - Neoplastic cell lines 24005
In vitro cellular and subcellular studies 32600

IT Major Concepts
Cell Biology; Enzymology (Biochemistry and Molecular Biophysics);
Metabolism

IT Miscellaneous Descriptors
VIMENTIN DESMIN GLIAL FIBRILLARY ACIDIC PROTEIN NEUROFILAMENT
TRIPLET PROTEINS CYTOKERATINS

ORGN Classifier
Suidae 85740
Super Taxa
Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Artiodactyls, Chordates, Mammals, Nonhuman Vertebrates,
Nonhuman Mammals, Vertebrates

RN 7440-70-2 (CALCIUM)
37353-41-6 (THIOL PROTEINASE)

ANSWER 4 OF 5 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

AN 1986:357972 BIOSIS

DN PREV198682062446; BA82:62446

TI EFFICIENT DEGRADATION IN-VITRO OF ALL INTERMEDIATE FILAMENT SUBUNIT
PROTEINS BY THE CALCIUM-ACTIVATED NEUTRAL THIOL
PROTEINASE FROM EHRLICH ASCITES TUMOR CELLS AND PORCINE KIDNEY.

AU VORGAS C E [Reprint author]; TRAUB P

CS MAX-PLANCK-INST ZELLBIOL, ROSENHOF, D-6802 LADENBURG/HEIDELBERG, FRG

SO Bioscience Reports, (1986) Vol. 6, No. 1, pp. 57-64.
CODEN: BRPTDT. ISSN: 0144-8463.

DT Article

FS BA

LA ENGLISH

ED Entered STN: 6 Sep 1986
Last Updated on STN: 6 Sep 1986

AB Vimentin, desmin, glial fibrillary acidic protein, neurofilament
triplet proteins, and a mixture of cytokeratins were digested with
Ca²⁺-activated neutral thiol proteinase
isolated from Ehrlich ascites tumor (EAT) cells and porcine kidney. All
intermediate filament proteins were degraded by the proteinase, although
with different rates and Ca²⁺ optima. These results are in part at
variance with our previous statement that the Ca²⁺-activated proteinase
from EAT cells is specific for vimentin and desmin.

CC Cytology - Animal 02506
Biochemistry studies - Minerals 10069
Enzymes - Physiological studies 10808
Metabolism - Proteins, peptides and amino acids 13012
Urinary system - Physiology and biochemistry 15504
Neoplasms - Neoplastic cell lines 24005
In vitro cellular and subcellular studies 32600

IT Major Concepts
Cell Biology; Enzymology (Biochemistry and Molecular Biophysics);
Metabolism

IT Miscellaneous Descriptors
VIMENTIN DESMIN GLIAL FIBRILLARY ACIDIC PROTEIN NEUROFILAMENT
TRIPLET PROTEINS CYTOKERATINS

ORGN Classifier
Suidae 85740
Super Taxa
Artiodactyla; Mammalia; Vertebrata; Chordata; Animalia
Taxa Notes
Animals, Artiodactyls, Chordates, Mammals, Nonhuman Vertebrates,
Nonhuman Mammals, Vertebrates

RN 7440-70-2 (CALCIUM)
37353-41-6 (THIOL PROTEINASE)

AN 1984:81710 CAPLUS

DN 100:81710

ED Entered STN: 12 May 1984

TI Large scale isolation, purification, and partial characterization of the intermediate filament-specific, calcium-activated proteinase from porcine kidney and Ehrlich ascites tumor cells: a comparative study

AU Traub, Peter

CS Max-Planck-Inst. Zellbiol., Ladenburg/Heidelberg, D-6802, Fed. Rep. Ger.

SO Archives of Biochemistry and Biophysics (1984), 228(1), 120-32

CODEN: ABBIA4; ISSN: 0003-9861

DT Journal

LA English

CC 7-2 (Enzymes)

AB The Ca^{2+} -activated, neutral thiol proteinase

specific for intermediate filament subunit proteins was isolated on a large scale from the postribosomal supernatant of a low-ionic-strength extract of porcine kidney and Ehrlich ascites tumor (EAT) cells. The purification

procedure encompassed DEAE-Sephacel ion exchange chromatog. of the material precipitating between 23 and 55% $(\text{NH}_4)_2\text{SO}_4$ saturation, followed by hydroxylapatite chromatog. and activated thiol-Sepharose 4B covalent chromatog. On the average, 25 mg of 62% pure enzyme was obtained from 500 g frozen kidney and 55 mg of 51% pure enzyme from 500 g EAT cells within a week. Both enzyme preps. were free of Ca^{2+} -independent proteolytic activities and indistinguishable with respect to their physicochem. and functional properties; their catalytic properties were indistinguishable from those of enzyme purified to homogeneity on arginine Me ester-Sepharose 4B. Because of this identity, porcine kidney proves to be an inexpensive source for the Ca^{2+} -activated proteinase which had previously been isolated and purified on a small scale from EAT cells (Nelson, W. J.; Traub, P., 1983). Despite a 38% protein contamination, the partially purified enzyme from porcine kidney is useful for the in vitro study of structure-function relationships of intermediate filaments and their subunit proteins. During affinity chromatog. of the partially purified proteinase from EAT cells on arginine Me ester-Sepharose 4B, a 100-kilodalton protein was purified which has a high affinity for arginine residues. It also occurs in porcine kidney, although at a considerably lower concentration. Its cellular localization and function remain to be determined

ST intermediate filament proteinase kidney carcinoma; Ehrlich ascites intermediate filament proteinase

IT Kidney, composition

(intermediate filament-specific proteinase of)

IT Proteins

RL: BIOL (Biological study)

(of intermediate filaments, proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)

IT Vimentins

RL: BIOL (Biological study)

(proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)

IT Carcinoma

(Ehrlich ascites, intermediate filament-specific proteinase of)

IT Proteins

RL: BIOL (Biological study)

(arginine-binding, of Ehrlich ascites cells, intermediate filament-specific proteinase in relation to)

IT Microfilament and Microtubule

(intermediate filament, proteins of, proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)

IT 78990-62-2

RL: BIOL (Biological study)

(of Ehrlich ascites cells and kidney, large-scale isolation and purification

AN 1984:81710 CAPLUS

DN 100:81710

ED Entered STN: 12 May 1984

TI Large scale isolation, purification, and partial characterization of the intermediate filament-specific, calcium-activated proteinase from porcine kidney and Ehrlich ascites tumor cells: a comparative study

AU Traub, Peter

CS Max-Planck-Inst. Zellbiol., Ladenburg/Heidelberg, D-6802, Fed. Rep. Ger.

SO Archives of Biochemistry and Biophysics (1984), 228(1), 120-32

CODEN: ABBIA4; ISSN: 0003-9861

DT Journal

LA English

CC 7-2 (Enzymes)

AB The Ca²⁺-activated, neutral thiol proteinase

specific for intermediate filament subunit proteins was isolated on a large scale from the postribosomal supernatant of a low-ionic-strength extract of porcine kidney and Ehrlich ascites tumor (EAT) cells. The purification

procedure encompassed DEAE-Sephacel ion exchange chromatog. of the material precipitating between 23 and 55% (NH₄)₂SO₄ saturation, followed by hydroxylapatite chromatog. and activated thiol-Sepharose 4B covalent chromatog. On the average, 25 mg of 62% pure enzyme was obtained from 500 g frozen kidney and 55 mg of 51% pure enzyme from 500 g EAT cells within a week. Both enzyme preps. were free of Ca²⁺-independent proteolytic activities and indistinguishable with respect to their physicochem. and functional properties; their catalytic properties were indistinguishable from those of enzyme purified to homogeneity on arginine Me ester-Sepharose 4B. Because of this identity, porcine kidney proves to be an inexpensive source for the Ca²⁺-activated proteinase which had previously been isolated and purified on a small scale from EAT cells (Nelson, W. J.; Traub, P., 1983). Despite a 38% protein contamination, the partially purified enzyme from porcine kidney is useful for the in vitro study of structure-function relationships of intermediate filaments and their subunit proteins. During affinity chromatog. of the partially purified proteinase from EAT cells on arginine Me ester-Sepharose 4B, a 100-kilodalton protein was purified which has a high affinity for arginine residues. It also occurs in porcine kidney, although at a considerably lower concentration. Its cellular localization and function remain to be

determined

ST intermediate filament proteinase kidney carcinoma; Ehrlich ascites intermediate filament proteinase

IT Kidney, composition

(intermediate filament-specific proteinase of)

IT Proteins

RL: BIOL (Biological study)

(of intermediate filaments, proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)

IT Vimentins

RL: BIOL (Biological study)

(proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)

IT Carcinoma

(Ehrlich ascites, intermediate filament-specific proteinase of)

IT Proteins

RL: BIOL (Biological study)

(arginine-binding, of Ehrlich ascites cells, intermediate filament-specific proteinase in relation to)

IT Microfilament and Microtubule

(intermediate filament, proteins of, proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)

IT 78990-62-2

RL: BIOL (Biological study)

(of Ehrlich ascites cells and kidney, large-scale isolation and purification

AN 1984:81710 CAPLUS

DN 100:81710

ED Entered STN: 12 May 1984

TI Large scale isolation, purification, and partial characterization of the intermediate filament-specific, calcium-activated proteinase from porcine kidney and Ehrlich ascites tumor cells: a comparative study

AU Traub, Peter

CS Max-Planck-Inst. Zellbiol., Ladenburg/Heidelberg, D-6802, Fed. Rep. Ger.

SO Archives of Biochemistry and Biophysics (1984), 228(1), 120-32

CODEN: ABBIA4; ISSN: 0003-9861

DT Journal

LA English

CC 7-2 (Enzymes)

AB The Ca²⁺-activated, neutral thiol proteinase

specific for intermediate filament subunit proteins was isolated on a large scale from the postribosomal supernatant of a low-ionic-strength extract of porcine kidney and Ehrlich ascites tumor (EAT) cells. The purification

procedure encompassed DEAE-Sephacel ion exchange chromatog. of the material precipitating between 23 and 55% (NH₄)₂SO₄ saturation, followed by hydroxylapatite chromatog. and activated thiol-Sepharose 4B covalent chromatog. On the average, 25 mg of 62% pure enzyme was obtained from 500 g frozen kidney and 55 mg of 51% pure enzyme from 500 g EAT cells within a week. Both enzyme preps. were free of Ca²⁺-independent proteolytic activities and indistinguishable with respect to their physicochem. and functional properties; their catalytic properties were indistinguishable from those of enzyme purified to homogeneity on arginine Me ester-Sepharose 4B. Because of this identity, porcine kidney proves to be an inexpensive source for the Ca²⁺-activated proteinase which had previously been isolated and purified on a small scale from EAT cells (Nelson, W. J.; Traub, P., 1983). Despite a 38% protein contamination, the partially purified enzyme from porcine kidney is useful for the in vitro study of structure-function relationships of intermediate filaments and their subunit proteins. During affinity chromatog. of the partially purified proteinase from EAT cells on arginine Me ester-Sepharose 4B, a 100-kilodalton protein was purified which has a high affinity for arginine residues. It also occurs in porcine kidney, although at a considerably lower concentration. Its cellular localization and function remain to be

determined

ST intermediate filament proteinase kidney carcinoma; Ehrlich ascites

intermediate filament proteinase

IT Kidney, composition

(intermediate filament-specific proteinase of)

IT Proteins

RL: BIOL (Biological study)

(of intermediate filaments, proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)

IT Vimentins

RL: BIOL (Biological study)

(proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)

IT Carcinoma

(Ehrlich ascites, intermediate filament-specific proteinase of)

IT Proteins

RL: BIOL (Biological study)

(arginine-binding, of Ehrlich ascites cells, intermediate filament-specific proteinase in relation to)

IT Microfilament and Microtubule

(intermediate filament, proteins of, proteinase of Ehrlich ascites cells and kidney specific for, purification and properties of)

IT 78990-62-2

RL: BIOL (Biological study)

(of Ehrlich ascites cells and kidney, large-scale isolation and purification

and properties of)

=>

and properties of)

=>

and properties of)

=>